Vibration during high frequency ventilation in neonates

Since there is no report quantifying vibration imposed upon neonates, we prospectively studied the vibration produced during high frequency ventilation (HFV) and compared it with that during conventional mechanical ventilation (CMV) of studied patients and weight matched controls (±250 g) receiving CMV or breathing spontaneously. A non-invasive accelerometer sensor (Analog Devices ADXL05, Norwood, MA, USA) was placed at the mid sternum or postauricular cranium to measure the linear vibration transmitted to the body and head, respectively (amplitude in time and frequency domains expressed in units of “g”).

From April to October 1998 we enrolled seven neonates treated with HFV (mean (SD) weight 2210 (1520) g, gestation 32 (7) weeks) and 14 weight matched controls (CMV group: n=7, 2100 (1730) g, 31 (8) weeks; spontaneous breathing group: n=7, 2230 (1520) g, 32 (7) weeks). The groups were not different with regard to body weight, length, and head circumference. Neonates received HFV at a frequency of 12 Hz, mean airway pressure of 14 (2) cm H₂O, amplitude of 39 (10) cm H₂O, and back up CMV at 6 breath/min. Higher amplitudes of vibration were detected during HFV than during CMV (0.098 (0.026) g vs 0.017 (0.006) g) at the chest and 0.011 (0.003) g vs 0.007 (0.001) g at the cranium, p<0.05) in six HFV treated neonates. One HFV treated neonate did not tolerate the switch to CMV. The vibrations at the chest and postauricular cranium in seven HFV treated neonates were higher than those of weight matched controls (fig 1, p<0.001), whereas no significant difference was found between the control groups. A higher amplitude of vibration at the chest was found in neonates with an adverse outcome than in normal survivors (0.136 (0.014) g vs 0.087 (0.024) g, respectively), while demographic data and the duration and amplitude of HFV were not different. Interestingly, the vibration at the chest exceeded the limit of whole body vibration in adults (0.05 g at 12.5 Hz third octave band for 24 hours per ISO 2631).

The significance of our observations is not known. While cardiovascular instability is commonly observed in neonates during HFV and has been related to a high lung volume ventilation strategy, cardiovascular effects of vibration have been reported in animal1 and clinical studies. Vibration during HFV may also contribute to the haemodynamic instability in neonates.

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<th>RMS vibration level (g)</th>
<th>HFV</th>
<th>CMV</th>
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Figure 1 Vibration in neonates during high frequency ventilation (HFV, n = 7), conventional mechanical ventilation (CMV, n = 7), and spontaneous breathing (SB, n = 7). (A) Representative vibration signals at the mid sternum of a neonate during HFV (left) and CMV (right). The upper panel shows the recorded time signal while the bottom panel displays the same information transformed to the frequency domain to display the dominant frequencies present in the signal. *p = 0.001 v HFV (ANOVA).
Furthermore, the effect of vibration on the developing brain is uncertain. We do not know whether the vibration will compromise the cerebral haemodynamic stability resulting in adverse neurological outcomes, especially in premature neonates who transmit vibration more efficaciously because of less body mass and fat compared with term neonates. Moreover, the combined effects of vibratory stress and environmental noise may contribute to hearing loss. Although no definitive vibration disease has been recognised in neonates, we have demonstrated the inadvertent exposure of neonates to excessive vibration. Research is required to examine the significance of HFV induced vibration and to reduce the vibration without compromising its effectiveness in critically ill neonates.

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National survey of detention and TB

In England and Wales the power to detain individuals with tuberculosis who pose a threat to public health lies principally in sections 37 and 38 of the Public Health Act 1984. Section 37 authorises a local authority of health to remove an individual to a suitable hospital and section 38 authorises the "detention for a prolonged period" (section 38 of the 1984 Public Health Act) to control tuberculosis. The duration of the period of detention varied from 3 days (n=1) to 6 months (n=6) with a median of 3 months. Thirty detention orders were issued during the 6 years surveyed; the year of issuance was given for 29. There was no apparent clustering in any health authority although 13 orders (43%) were issued in London. The duration of the period of the detention orders varied from 3 days (n=1) to 6 months (n=6) with a median of 3 months.

The number of detention orders being issued for individuals with tuberculosis since 1994 has increased significantly (p<0.005). By 1999 0.2% of individuals notified with pulmonary tuberculosis were issued with detention orders. Regressions analysis, taking into account health authority response rates and notification rates for pulmonary tuberculosis, shows a significant increase in the issuance of detention orders since 1994 (b=1.8283, p=0.9; fig 1).

The reasons for the increase in numbers of detention orders being issued are unclear. Increases in the incidence of tuberculosis outbreaks in healthcare settings and the scourge of drug resistant and multidrug resistant strains have over the past decade, perhaps concentrated the minds of clinicians and public health physicians on ensuring that patients comply with treatment, and this may be playing a part. Elsewhere, notably in New York but also in Europe, consideration of the legal and ethical aspects of contemporary control measures has resulted in legislative amendments to public health laws which have enabled public health authorities to detain, for prolonged periods, patients with tuberculosis who will not or cannot comply with treatment.11 Failures in tuberculosis control allied to insufficient resources to facilitate patients' adherence to treatment, particularly in London, may also be contributing to the use of more restrictive measures by the authorities.12 An alternative explanation may be that, because of drug resistance and associated HIV infection, the treatment of tuberculosis is becoming increasingly complex, demanding greater commitment from patients and clinicians. This survey suggests that there is a need to monitor formally, in an ongoing fashion, trends in the issuance of detention orders for individuals with tuberculosis.

I thank the consultants in communicable disease control in England and Wales for assisting with this research and the Communicable Disease Surveillance Centre for providing the data on pulmonary tuberculosis notifications.

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National survey of detention and TB

In England and Wales the power to detain individuals with tuberculosis who pose a threat to public health lies principally in sections 37 and 38 of the Public Health Act 1984. Section 37 authorises a local authority officer to remove an individual to a suitable hospital and section 38 authorises the “detention for a period specified in the order”. By way of the Public Health (Infectious Diseases) Regulations 1998, in addition to the five notifiable diseases (cholera, plague, relapsing fever, smallpox, typhus), these sections currently apply to tuberculosis of the respiratory tract in an infectious state (although the term “infectious” is not defined by law).

Because central records are not kept, it has been unclear how many individuals are detained each year under legislation. No research has been conducted in Britain to determine trends in the use of detention as a public health tool in the control of tuberculosis, although a survey conducted in the early 1990s of consultants in communicable diseases and medical officers of environmental health (CCDC/MOEH) reported the issuance of six orders, “equivalent to less than one use of the sections for every hundred years of CCDC/MOEH experience”. A brief structured postal questionnaire was sent to consultants in communicable disease control in all 99 health authorities in England and Wales in February 2000 requesting information on whether any detention orders (section 38 of the 1984 Public Health Act) for individuals with tuberculosis had been issued in the health authority to which the letter had been sent since 1993. Information was received from consultants in 97 of the health authorities.

Thirtynine detention orders were issued during the 6 years surveyed; the year of issuance was given for 29. There was no apparent clustering in any health authority although 13 orders (43%) were issued in London. The duration of the period of the detention orders varied from 3 days (n=1) to 6 months (n=6) with a median of 3 months.

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the general population. The mean age of onset was 57 years, significantly older than the 40 years reported in patients from tertiary centres and quoted by Conron and Beynon. We found there was a slight male preponderance (65%), also shown in many previous studies. The commonest sites of extrarespiratory involvement were, in descending frequency, the nervous system, joints and muscles, kidneys, skin, heart, and gastrointestinal tract. Involvement of the gastrointestinal tract occurred in only 30% and abdominal pain was uncommon. Cardiac involvement occurred in 44% and was the cause of death in two. Renal involvement, although occurring in nearly half the patients, was usually mild and none had severe renal failure.

The mean age of onset of asthma was 50 years and, although not reported, the asthma was not usually troublesome or severe when the silicosis developed. Indeed, very few patients were on oral steroids for their asthma at that time. Very rarely, patients present with CSS who do not have asthma. In our series there was one such patient who satisfied the original histological features of Churg and Strauss.

We found that the criteria developed by Lanham et al were the most useful, particularly an eosinophil count of ≥1.5 × 10⁹/l. While this is arbitrary, only one of our patients had a level below this and that was 1.4 × 10⁹/l.

Acute rib fracture pain in CF

Recent papers in Thorax have described the high prevalence of low bone mineral density (BMD) in individuals with cystic fibrosis; these patients are at increased risk of fractures. Rib fracture pain can often be difficult to treat, despite standard analgesia such as non-steroidal anti-inflammatory drugs and opiates. Rib pain can impair sputum clearance and lead to an exacerbation of CF pulmonary disease. There are reports that calcitonin can relieve bone pain for patients with osteoporotic vertebral fractures. Recently, we have successfully used subcutaneous calcitonin for the treatment of rib fracture pain in two patients with CF.

A 25 year old woman fractured two ribs when she was crushed in the crowd at a rock concert and a 28 year old man fractured ribs when she was crushed in the crowd at a rock concert. Both patients were taking oral prednisolone at a dose of 10 mg per day. The female patient was taking paracetamol 1 g qds and morphine sulphate modified release 30 mg bd; the male patient was taking paracetamol 1 g qds and ibuprofen 600 mg tds. Both patients were given courses of antibiotics as the pain was leading to an exacerbation of their CF lung disease. Subcutaneous calcitonin (100 IU) was given in a dose of 50 units once daily. The pain completely resolved within 48 hours in both cases, and the patients were able to mobilise, perform sputum clearance, the other analgesics were withdrawn, and the chest exacerbations resolved. The calcitonin injections were continued for a total of 7 days, then stopped without recurrence of any pain. Neither patient experienced any side effects from the calcitonin.

Although calcitonin is involved in the regulation of bone turnover, the mechanism of its analgesic action is unknown. It reduces bone resorption and bone turnover but may also have central analgesic effects. Conversely, intravenous bispophosphonates, given to improve bone density, were associated with severe bone pain in individuals with CF.

Calcitonin has been shown effective in patients with CF and rib fractures if adequate sputum clearance is to be achieved and an acute deterioration in lung disease avoided. Calcitonin should be considered as an analgesic in this situation. Settling sputum may reduce morbidity and mortality associated with rib fractures in this group of patients.

We therefore think that increased sputum ET-1 levels found in patients with COPD during an acute exacerbation could represent a true increase in local ET production in patch with a firm relationship between venous and sputum ET-1 levels could not be established.

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CF and antistaphylococcal prophylaxis

Dr Robinson’s review of cystic fibrosis (CF) touches on the use of continuous anti-staphylococcal antibiotic prophylaxis. Dr Robinson reiterates the oft repeated assertion that there is an association between the use of prophylactic antibiotics and the early acquisition of pulmonary infection with Pseudomonas aeruginosa in patients with CF. This association has entered CF folklore but is not supported by any published evidence. The paper cited by Robinson describes a pilot study of cephalexin which lasted 2 months. A subsequent multicentre, randomised, placebo-controlled trial of cephalexin, commenced in children under 2 years, has been undertaken and its methodology described. However, no results have so far appeared in the published literature.

We have recently published an updated systematic review of randomised controlled trials of prophylactic antibiotics in CF. This describes data from three studies involving 189 children. We found that fewer children...
who received prophylaxis from birth had one or more isolates of *Staphylococcus aureus* over a 3 year period. There was also evidence at 2 years that less time was spent in hospital in the prophylaxis group. The number of children receiving prophylaxis who had one or more isolates of *P aeruginosa* over a 3 year period was half that of the control group who had intermittent antibiotic treatment only. This was not, however, statistically significant (Peto odds ratio 0.54, 95% confidence interval 0.23 to 1.26).

The steering group of the North American cephalixin trial have indicated that its results will be published soon (Eliezer Nussbaum, personal communication). However, until there is published evidence from at least one properly designed randomised controlled trial, the proposal that prophylaxis encourages pulmonary infection with *P aeruginosa* remains entirely speculative.

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**NOTICES**

**The Dr H M (Bill) Foreman Memorial Fund**

The Trustees of the above fund invite applications for grants relating to study in respiratory disease and allied fields. Limited funds are available for registered medical practitioners to assist in travelling to countries other than their own to study respiratory disease, and also for support for clinical research abroad.

The HMF Fund has been able to fund two medical student projects in the last 18 months, one on TB in Malaysia and one on TB in Ghana, and has awarded three travel grants to study the following aspects of respiratory disease: Dr Veronika White (London) to study TB in Bangladesh; Dr R T Jagoe (Newcastle upon Tyne) to study the ATP-ubiquitin-proteasome proteolytic system in Boston, USA; Dr J S Parmar (Cambridge) to study cell motility in Toronto; and a grant to Dr Anne Chang (Brisbane) to study the relationship between cough and asthma.

Intending applicants should write for further details to Dr Brian H Davies, Llandough Hospital, Penarth, Vale of Glamorgan CF64 2AA, UK.

**Pharmacology of Asthma**

A course on the “Pharmacology of Asthma” organised by Professor Peter Barnes will be held at the Imperial College School of Medicine at the National Heart & Lung Institute in collaboration with the Royal Brompton Hospital, Dovehouse Street, London SW3 6LY, UK on 26–29 November 2001. The course is suitable for physicians or scientists with an interest in the pharmacology and therapeutics of asthma. For further information please contact the Postgraduate Education Centre, Imperial College School of Medicine at the National Heart & Lung Institute, Dovehouse Street, London SW3 6LY. Telephone: 020 7351 8172. Fax: 020 7351 8246. Email: shortcourses.nhli@ic.ac.uk

**Respiratory Medicine**

A conference on Respiratory Medicine will be held at the Royal College of Physicians of Edinburgh on 26 October 2001. For further information contact Ms Eileen Strawn, Symposium Coordinator. Telephone 0131 225 7324. Fax 0131 220 4593. Email: e.strawn@rcpe.ac.uk. Website: www.rcpe.ac.uk.